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NEWS 16	APR 27	NLDB: New search and display fields available

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FILE LAST UPDATED: 7 May 2004 (20040507/ED)

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         17760 SOMATOSTATIN?
        474147 MODUL?
L1        1496 SOMATOSTATIN? AND MODUL?

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         90772 COMPLICAT?
           630 DIABETES (W) COMPLICAT?
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         30096 COMPLICATION?
          1772 DIABETE? (4W) COMPLICATION?
L3           0 L1 AND DIABETE? (4W) COMPLICATION?

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L4          41 L1 AND DIABETE?

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L5           0 L4 AND DT/REVIEW

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L6           5 L4 AND REVIEW/DT

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Full Text | Citing References

ACCESSION NUMBER: 2003:246328 HCAPLUS
DOCUMENT NUMBER: 139:270068
TITLE: Pharmacological agents that directly **modulate** insulin secretion
AUTHOR(S): Doyle, Maire E.; Egan, Josephine M.
CORPORATE SOURCE: Diabetes Section, National Institute on Aging, National Institutes of Health, Baltimore, MD, USA
SOURCE: Pharmacological Reviews (2003), 55(1), 105-131
CODEN: PAREAQ; ISSN: 0031-6997
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal; **General Review**

LANGUAGE: English

AB A review. Blood glucose levels are sensed and controlled by the release of hormones from the islets of Langerhans in the pancreas. The β-cell, the insulin-secreting cell in the islet, can detect subtle increases in circulating glucose levels and a cascade of mol. events spanning the initial depolarization of the β-cell membrane culminates in exocytosis and optimal insulin secretion. Here we review these processes in the context of pharmacol. agents that have been shown to directly interact with any stage of insulin secretion. Drugs that **modulate** insulin secretion do so by opening the KATP channels, by interacting with cell-surface receptors, by altering second-messenger responses, by disrupting the β-cell cytoskeletal framework, by influencing the mol. reactions at the stages of transcription and translation of insulin, and/or by perturbing exocytosis of the insulin secretory vesicles. Drugs acting primarily at the KATP channels are the sulfonylureas, the benzoic acid derivs., the imidazolines, and the quinolines, which are channel openers, and finally diazoxide, which closes these channels. Methylxanthines also work at the cell membrane level by antagonizing the purinergic receptors and thus increase insulin secretion. Other drugs have effects at multiple levels, such as the calcineurin inhibitors and **somatostatin**. Some drugs used extensively in research, e.g., colchicine, which is used to study vesicular transport, have no effect at the pharmacol. doses used in clin. practice. We also briefly discuss those drugs that have been shown to disrupt β-cell function in a clin. setting but for which there is scant information on their mechanism of action.

REFERENCE COUNT: 300 THERE ARE 300 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 5 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1998:213707 HCAPLUS

DOCUMENT NUMBER: 128:279034

TITLE: Hypothalamic and hypophyseal regulation of growth hormone secretion

AUTHOR(S): Bluet-Pajot, Marie-Therese; Epelbaum, Jacques; Gourdji, Daniele; Hammond, Constance; Kordon, Claude

CORPORATE SOURCE: Unite de Recherche sur la Dynamique des Systemes Neuroendocriniens (U159), INSERM, Paris, 75014, Fr.

SOURCE: Cellular and Molecular Neurobiology (1998), 18(1), 101-123
CODEN: CMNEDI; ISSN: 0272-4340

PUBLISHER: Plenum Publishing Corp.

DOCUMENT TYPE: Journal; **General Review**

LANGUAGE: English

AB A review, with ~150 refs. Regulation of pulsatile secretion of growth hormone (GH) relies on hypothalamic neuronal loops, major transmitters involved in their operation are growth hormone releasing hormone (GHRH) synthesized mostly in arcuate nucleus (ARC) neurons, and **somatostatin** (SRIH), synthesized both in hypothalamus periventricular (PVe) and ARC neurons. Neurons synthesizing both peptides can inhibit each other in a reciprocal manner. Other neuropeptides synthesized in ARC neurons, such as galanin, or in ARC interneurons, such as neuropeptide Y (NPY), are able to **modulate** synthesis and release of GHRH and SRIH into the hypothalamohypophyseal portal system. In addn., the hitherto uncharacterized endogenous ligand of the recently cloned growth hormone releasing peptide receptor, expressed mostly in the ARC, triggers GH

release, presumably by actions on ARC interneurons. Thyroid, gonadal, and adrenal steroid hormones also affect the GHRH-SRIH balance; a differential distribution of sex steroid receptors in the ARC and the PVe is likely to account for the different pattern of GH secretion in male and female animals. Growth hormone itself is able to inhibit the amplitude of GH secretory episodes and to increase their frequency, by entering the brain (presumably by receptor-mediated internalization at the level of the choroid plexus) and acting subsequently on ARC neurons. At the pituitary level, major neurotransmitters regulating GH cells act on receptors of the VIP/PACAP/GHRH family and of the **somatostatin** family, in particular, sst2 and sst3. Those are coupled to accumulation of cAMP as a second messenger. In addn., patch-clamp expts. and measurement of intracellular Ca2+ indicate that GH cells present characteristics, GHRH-dependent, but self-maintained Ca2+ spikes and [Ca2+]i transients, which reflect adaptive mechanisms to constraints of episodic release. Recent data on transcription factors affecting GH gene expression and somatotrope differentiation are also summarized. Regulation and differentiation of somatotropes also depend upon paracrine processes within the pituitary itself and involve growth factors and several neuropeptides, for instance, vasoactive intestinal peptide, angiotensin 2, endothelin, and activin. Finally, characteristic changes occur in the GH secretory pattern under discrete, pathol. conditions, such as abnormal growth and dwarfism, **diabetes**, and acromegaly, as well as during inflammatory processes.

REFERENCE COUNT: 149 THERE ARE 149 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1998:126168 HCAPLUS
DOCUMENT NUMBER: 128:266280
TITLE: Cellular regulation of islet hormone secretion by the incretin hormone glucagon-like peptide 1
AUTHOR(S): Gromada, J.; Holst, Jens Juul; Rorsman, Patrik
CORPORATE SOURCE: The Symbion Science Park, Novo Nordisk A/S, Department of Islet Cell Physiology, Fruebjergvej 3, Copenhagen, DK-2100, Den.
SOURCE: Pfluegers Archiv (1998), 435(5), 583-594
CODEN: PFLABK; ISSN: 0031-6768
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 86 refs. Glucagon-like peptide 1 is a gastrointestinally derived hormone with profound effects on nutrient-induced pancreatic hormone release. GLP-1 **modulates** insulin, glucagon and **somatostatin** secretion by binding to guanine nucleotide binding protein-coupled receptors resulting in the activation of adenylate cyclase and generation of cAMP. In the B-cell, cAMP, via activation of protein kinase A, interacts with a plethora of signal transduction processes including ion channel activity, intracellular Ca2+ handling and exocytosis of the insulin-contg. granules. The stimulatory action of GLP-1 on insulin secretion, contrary to that of the currently used hypoglycemic sulfonylureas, is glucose dependent and requires the presence of normal or elevated concns. of the sugar. For this reason, GLP-1 attracts much interest as a possible novel principle for the treatment of human type-2 **diabetes**. Here the authors review the actions of GLP-1 on islet cell function and attempt to integrate current knowledge into a working model for the control of pancreatic hormone secretion.

REFERENCE COUNT: 86 THERE ARE 86 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 5 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1996:93560 HCAPLUS
DOCUMENT NUMBER: 124:171923
TITLE: The role of insulin-like growth factors (IGFs) and IGF binding proteins in diabetic nephropathy
AUTHOR(S): Flyvbjerg, A.; Landau, D.; Kiess, W.; Bondy, C.; Chin, E.; Raz, I.; Phillip, M.; LeRoith, D.
CORPORATE SOURCE: Institute Experimental Clinical Research, Aarhus, DK-8000, Den.
SOURCE: International Congress Series (1995), 1100(Diabetes 1994), 345-9
CODEN: EXMDA4; ISSN: 0531-5131
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 28 refs. Diabetic kidney disease is characterized by an early increase in kidney size, glomerular vol. and kidney function and later by the development of mesangial proliferation, accumulation of glomerular extracellular matrix and increased urinary albumin excretion (UAE). Insulin-like growth factors (IGFs) have a long and distinguished history in **diabetes** mellitus with possible participation in the development of long-term complications. In exptl. **diabetes**, increased renal size and function is preceded by a rise in the renal IGF-I, IGF binding proteins (IGFBPs), and IGF-II/mannose-6-phosphate receptor (IGF-II/Man-6-P receptor) concn. Diabetic dwarf rats with isolated growth hormone (GH) and IGF-I deficiency exhibit slower and lesser initial renal and glomerular hypertrophy than diabetic controls with intact pituitary, indicating that IGF-I and GH may be involved in the **modulation** of renal enlargement. Strict insulin treatment or administration of a **somatostatin** analog (octreotide) equally inhibits kidney IGF-I accumulation and growth, the latter without affecting blood glucose levels. These results indicate that IGFs act as initiating growth factors for the diabetic renal growth in short-term exptl. **diabetes**. Whether the IGF system is also involved in long-term diabetic renal changes is unknown. However, 6-mo administration of octreotide to diabetic rats reduces diabetic UAE, renal and glomerular hypertrophy without affecting metabolic control. Furthermore, long-term diabetic dwarf rats, with a **diabetes** duration of 6 mo, display a smaller degree of renal and glomerular hypertrophy and rise in UAE, when compared to pituitary intact diabetic rats. Finally, specific changes occur in the renal IGFBP mRNA expression in long-term **diabetes**. In conclusion, the cited studies suggest that a complex IGF system comprising IGF ligands, IGF receptors, and IGFBPs may be involved in the development of the diabetic kidney disease.

L6 ANSWER 5 OF 5 HCAPLUS COPYRIGHT 2004 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1995:906675 HCAPLUS
DOCUMENT NUMBER: 123:306680
TITLE: Intervention in diabetic vascular disease by **modulation** of growth factors
AUTHOR(S): Serri, Omar; Renier, Genevieve
CORPORATE SOURCE: Metabolic Unit, Notre-Dame Hospital, Montreal, QC, Can.
SOURCE: Metabolism, Clinical and Experimental (1995), 44(10,

Suppl. 4), 83-90
CODEN: METAAJ; ISSN: 0026-0495
PUBLISHER: Saunders
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review, with 106 refs. Several growth factors have been implicated in the derangements of cellular metab. and proliferation that occur in **diabetes**, e.g., kidney mesangial expansion, retinal neovascular formation, and acceleration of atherosclerosis in large vessels. These phenomena contribute to the development and progression of diabetic microvascular and macrovascular disease. Pharmacol. interventions aimed at reducing growth factor alterations, among other actions in diabetic vasculopathy, include a multitude of classes of drugs, such as angiotensin-converting enzyme (ACE) inhibitors, calcium antagonists, lipid-lowering drugs, and **somatostatin** analogs. New potential interventions, ie, antisense oligonucleotide local delivery, are being applied in growth factor research and may prove beneficial in diabetic macrovascular disease.

=>

Table of Contents

Protective Groups In Organic Synthesis

Greene Et. al.

5. J. W. Barton, "Protection of N-H Bonds and NR_3," in *Protective Groups in Organic Chemistry*, J. F. W. McOmie, Ed., Plenum Press, New York and London, 1973, pp. 46–56.

6. Y. Wolman, "Protection of the Amino Group," in *The Chemistry of the Amino Group*, S. Patai, Ed., Wiley-Interscience, New York, 1968, Vol. 4, pp. 669–682.

7. E. Gross and J. Meienhofer, Eds., *The Peptides: Analysis: Synthesis, Biology*, Vol. 3: *Protection of Functional Groups in Peptide Synthesis*, Academic Press, New York, 1981.

Formamide:
R_2NCHO (Chart 9)

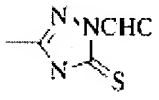
Formation

1. 98% HCO_2H, Ac_2O, 25°, 1 h, 78–90% yield.[1,2] The use of formic acetic anhydride for esterification and amide formation has been reviewed.[3]

2. HCO_2H, DCC, Pyr, 0°, 4 h, 87–90% yield.[4] These conditions produce *N*-formyl derivatives of *t*-butyl amino acid esters with a minimum of racemization.

3. HCO_2H, $EtN{=}C{=}N(CH_2)_3NMe_2{\cdot}HCl$, 0°, 15 min; then *N*-methylmorpholine, 5°, 20 h, 65–96% yield. This method can be used with amine hydrochlorides.[5]

4. C_6F_5OCHO, $CHCl_3$, rt, 5–30 min, 85–99% yield.[6]

5.  This reagent also formylates alcohols in the presence of added base.[7]

6. *t*-$BuMe_2SiCl$, DMAP, Et_3N, DMF, 35–60°, 65–85% yield.[8]

7. DMF, silica gel, heat, 5 h, 100% yield,[9] or DMF, ZrO, heat, 5 h, 92% yield.[10]

8. HCO_2Et, heat.[11]

9. Triethyl orthoformate, 50–100% yield.[12]

10. HCO_2CH_2CN, CH_2Cl_2, rt, 12 h, 62–97% yield.[13]

11. Vinyl formates readily react with amines, alcohols, and phenols to give the formamide or ester.[14]

Cleavage

1. HCl, H_2O, dioxane, 25°, 48 h, or reflux, 1 h, 80–95% yield.[1]

2. Hydrazine, EtOH, 60°, 4 h, 60–80% yield.[15]

3. H_2/Pd-C, THF, HCl, 25°, 5–7 h, quant.[16]

4. 15% H_2O_2, H_2O, 60°, 2 h, 80% yield.[17]

5. AcCl, $PhCH_2OH$, 20°, 24 h, or 60°, 3 h, good yields.[18]

Special -NH Protective Groups

N-*Alkyl and* N-*Aryl Amines*

N-Methylamine:
CH_3NR_2

Formation

1. Methylamines are commonly formed by reacting the amine with a methylating agent such as MeI or dimethyl sulfate.

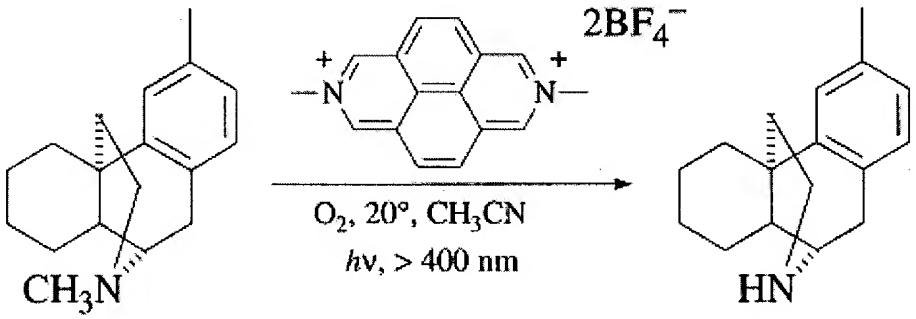
2. Preparation from an amine and $TMSCHN_2$ (HBF_4, CH_2Cl_2, H_2O) has also been explored.

3. For primary aromatic amines: dimethyl carbonate, Y-Zeolite, 130–150°, 72–93% yields.[1]

4. HCHO, HCO_2H, 5° then reflux, 12 h, 91% yield.[2,3]

Cleavage

1. The cleavage of a methylamine can be accomplished photochemically in the presence of an electron acceptor such as 9,10-dicyanoanthracene.[4]



2. Photolysis with visible light, DAP^{+2}; TMSCN. The photochemical reaction generates an iminium ion that is trapped with cyanide.[5]

3. CH_2=CHOCOCl, K_2CO_3, CH_2Cl_2.[6] The *N*-methyl group of a tertiary amine is converted to a vinyl carbamate that is easily hydrolyzed.

4. I_2, CaO, THF, MeOH. A dimethylaniline is converted to a monomethylaniline.[7]

5. CS_2, MeI, THF, 6 h, 30°, 97% yield. *N*-Methylpiperidine is converted to a dithiocarbamate.[8]

6.

Ref. 9

7. *t*-BuOOH, $RuCl_2(Ph_3P)_2$, benzene, rt, 3 h, 83% yield. The methyl group is

converted to *t*-$BuOOCH_2NR_2$, which can then be hydrolyzed, releasing the secondary amine.[10] The oxidation of amines has been reviewed.[11]

8. PhSeH, 160°, 5 days, 68% yield.[12]

9. $RuCl_3$, H_2O_2, MeOH, 55–80% yield.[13] These conditions convert the methyl to a MOM group that can be removed by hydrolysis.

1. M. Selva, A. Bomben, and P. Tundo, *J. Chem. Soc., Perkin Trans. 1*, 1041 (1997).

2. G. Chelucci, M. Falorni, and G. Giacomelli, *Synthesis*, 1121 (1990).

3. For a review of the Leukart reaction, see M. L. Moore, *Org. React.*, **5**, 301 (1949).

4. J. Santamaria, R. Ouchabane, and J. Rigaudy, *Tetrahedron Lett.*, **30**, 2927 (1989).

5. J. Santamaria, M. T. Kaddachi, and J. Rigaudy, *Tetrahedron Lett.*, **31**, 4735 (1990).

6. J. R. Ferguson, K. W. Lumbard, F. Scheinmann, A. V. Stachulski, P. Stjernlöf, and S. Sundell, *Tetrahedron Lett.*, **36**, 8867 (1995); R. A. Olofson, R. C. Schnur, L. Bunes, and J. P. Pepe, *ibid.*, 1567 (1977).

7. K. Acosta, J. W. Cessac, P. N. Rao, and H. K. Kim, *J. Chem. Soc., Chem. Commun.*, 1985 (1994).

8. M. D. Pujol and G. Guillaumet, *Synth. Commun.*, **22**, 1231

9. J. P. Gesson, J. C. Jacquesy, and M. Mondon, *Synlett*, 669 (1990).

10. S.-I. Murahashi, T. Naota, and K. Yonemura, *J. Am. Chem. Soc.*, **110**, 8256 (1988).

11. S.-I. Murahashi, *Angew. Chem., Int. Ed. Engl.*, **34**, 2443 (1995).

12. R. P. Polniaszek and L. W. Dillard, *J. Org. Chem.*, **57**, 4103 (1992).

13. S.-I. Murahashi, T. Naota, N. Miyaguchi, and T. Nakato, *Tetrahedron Lett.*, **33**, 6991 (1992).

_N_-*t*-Butylamine:
$(CH_3)_3CNR_2$

The *t*-butyl group can be cleaved from a cyclopropylamine upon prolonged heating in acid (H_3O^+, reflux, 3–5 days).[1]

1. N. De Kimpe, P. Sulmon, and P. Brunet, *J. Org. Chem.*, **55**, 5777 (1990).

_N_-Allylamine:
CH_2=$CHCH_2NR_2$ (Chart 10)

Formation

1. Allyl chloride, Cu(0), $Cu(ClO_4)_2 \cdot 6H_2O$, Et_2O, 97% yield.[1]

2. AllylOAc, $Pd(Ph_3P)_4$, diisopropylamine.[2]

3. $Ni(cod)_2$, $Bu_4N^+PF_{6^-}$, dppb, THF, 50°.[3]

4. Allyl bromide, K_2CO_3, THF, heat, 75% yield.[4] This is a fairly general method that has been used widely for the preparation of allylamines.

Cleavage

1. Isomerization to the enamine (*t*-BuOK, DMSO), followed by hydrolysis.[5]

2. Rhodium-catalyzed isomerization.[6] Ru(cod)(cot) has been used to convert an allylamine into an enamine.[7]

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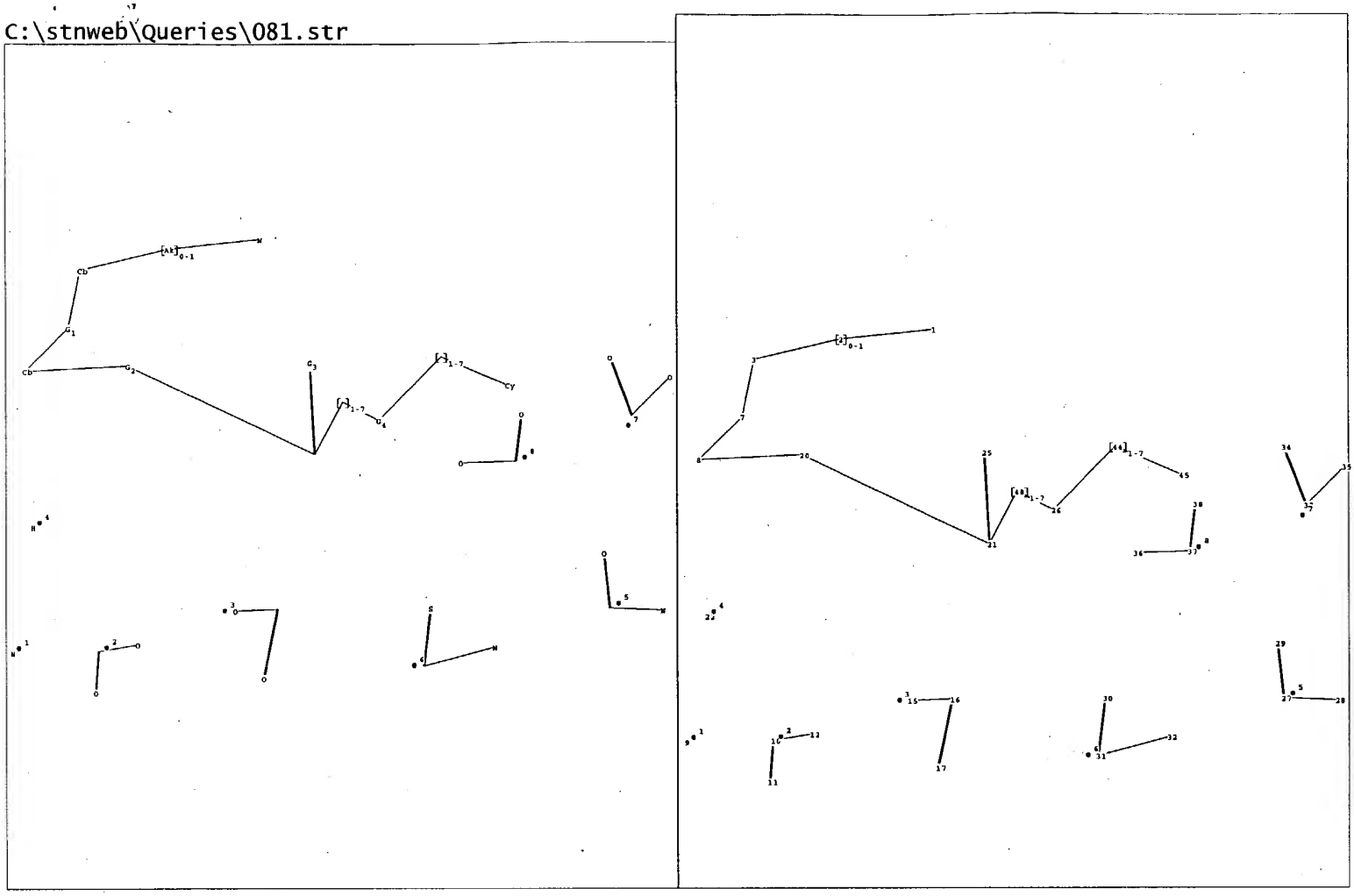

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chain bonds :
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27-29 27-28 30-31 31-32 33-34 33-35 36-37 37-38 44-45
exact/norm bonds :
1-2 2-3 3-7 7-8 8-20 10-12 10-11 15-16 16-17 20-21 21-25 26-44 26-48 27-29
27-28 30-31 31-32 33-34 33-35 36-37 37-38 44-45
exact bonds :
21-48

G1:O,S

G2:[*1],[*2],[*3]

G3:[*1],[*2],[*3],[*4]

G4:[*5],[*6],[*7],[*8]

Match level :
1:CLASS 2:CLASS 3:Atom 7:CLASS 8:Atom 9:CLASS 10:CLASS 11:CLASS 12:CLASS 15:CLASS
16:CLASS 17:CLASS 20:CLASS 21:CLASS 22:CLASS 25:CLASS 26:CLASS 27:CLASS 28:CLASS
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38:CLASS 44:CLASS 45:Atom 48:CLASS

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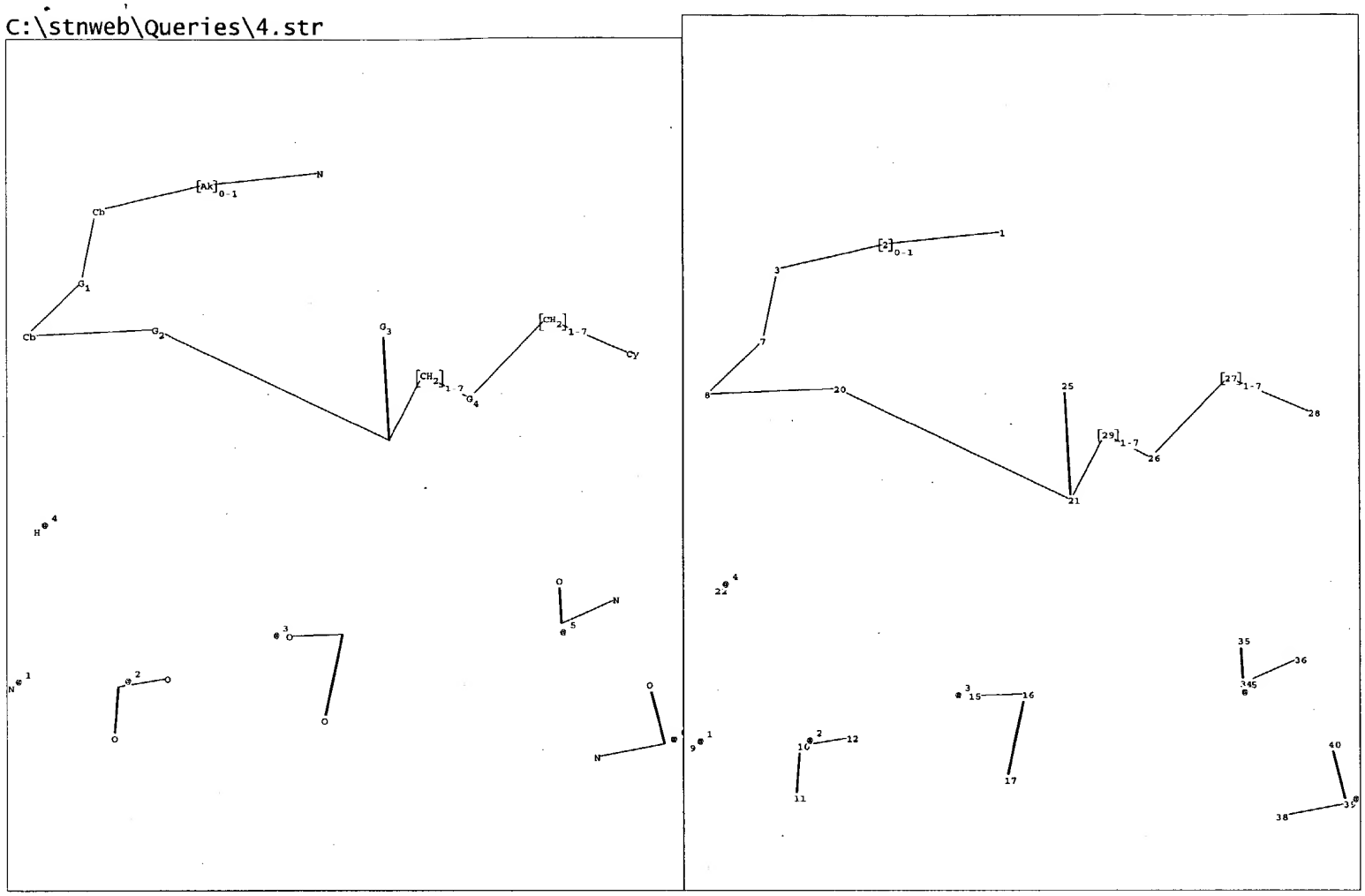

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39 40
chain bonds :
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27-28 34-35 34-36 38-39 39-40
exact/norm bonds :
1-2 2-3 3-7 7-8 8-20 10-12 10-11 15-16 16-17 20-21 21-25 26-27 26-29 27-28
34-35 34-36 38-39 39-40
exact bonds :
21-29

G1:O,S

G2:[*1],[*2],[*3]

G3:[*1],[*2],[*3],[*4]

G4:[*5],[*6]

Match level :
1:CLASS 2:CLASS 3:Atom 7:CLASS 8:Atom 9:CLASS 10:CLASS 11:CLASS 12:CLASS 15:CLASS
16:CLASS 17:CLASS 20:CLASS 21:CLASS 22:CLASS 25:CLASS 26:CLASS 27:CLASS 28:Atom
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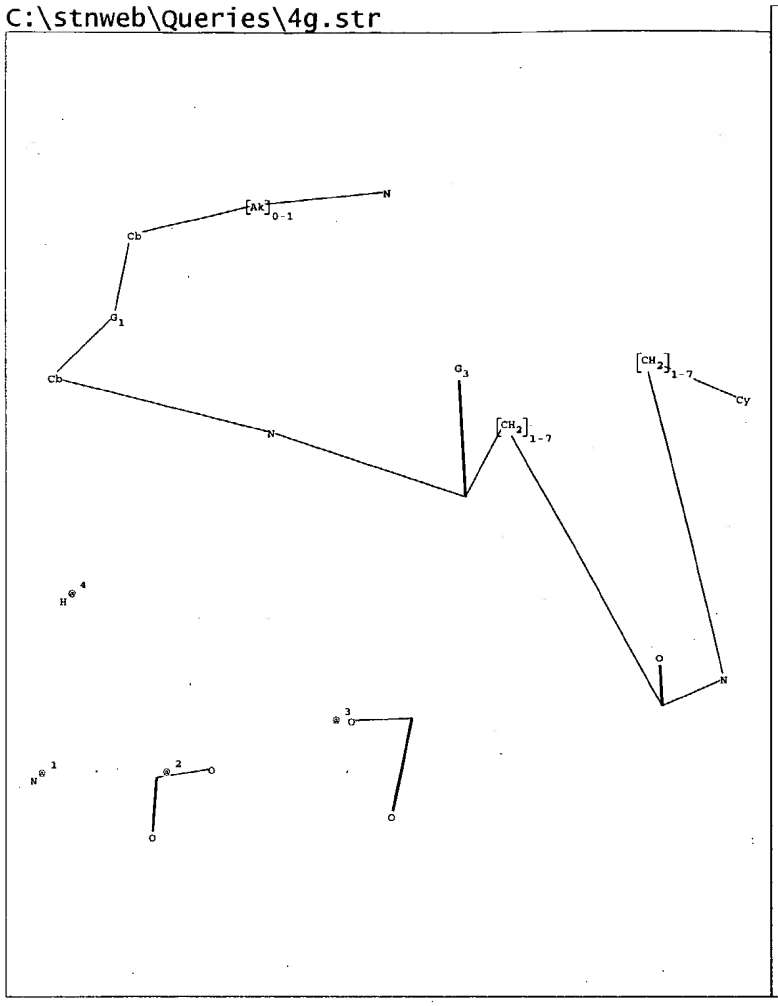

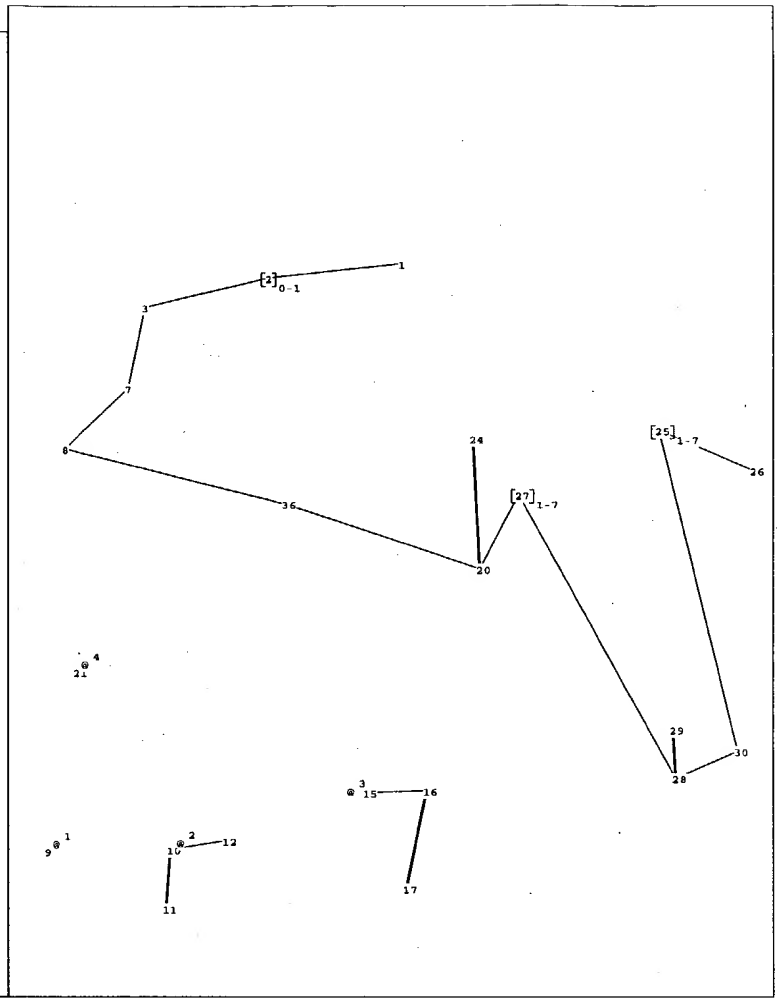

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chain bonds :
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27-28 28-29 28-30
exact/norm bonds :
1-2 2-3 3-7 7-8 10-12 10-11 15-16 16-17 20-24 25-26 28-29 28-30
exact bonds :
8-36 20-27 20-36 25-30 27-28

G1:O,S

G2:[*1],[*2],[*3]

G3:[*1],[*2],[*3],[*4]

G4

Match level :
1:CLASS 2:CLASS 3:Atom 7:CLASS 8:Atom 9:CLASS 10:CLASS 11:CLASS 12:CLASS 15:CLASS
16:CLASS 17:CLASS 20:CLASS 21:CLASS 24:CLASS 25:CLASS 26:Atom 27:CLASS 28:CLASS
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